Postoperative Deep Surgical-Site Infection after Instrumented Spinal Surgery: A Multicenter Study

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Abstract

A retrospective survey revealed 37 cases (1.1%) of deep surgical-site infection (SSI) among 3,462 instrumented spinal surgeries between 2004 and 2008. Excluding 8 patients who were unclassifiable, we categorized 29 patients into 3 groups of similar backgrounds—thoracolumbar degenerative disease (the DEG group; $n = 15$), osteoporotic vertebral collapse (the OVC group; $n = 10$), and cervical disorders (the cervical group; $n = 4$)—and investigated the key to implant salvage. Final respective implant retention rates for the groups were 40, 0, and 100%, with the OVC group having the worst rate ($p < 0.01$). In the DEG group with early infection, those whose implants were retained had lower body temperatures, lower white blood cell counts, and a lower rate of discharge at the time of SSI diagnosis ($p < 0.05$). Implant retention may be affected by initial spinal pathology. In the DEG group, debridement before drainage may be advantageous to implant salvage.

Keywords
► surgical-site infection
► spinal surgery
► spinal instrumentation
► treatment

The incidence of surgical-site infection (SSI) after instrumented spinal surgery has been reported to range from 2.2 to 8.5%.[1–8] Although prevention of SSI has been emphasized and is practiced, SSI has not been eradicated, and the treatment of deep infected wounds, especially with instrumentation, is one of the most complicated problems in spinal surgery. The goal of SSI treatment after instrumented spinal surgery is to resolve infection as well as to maintain spinal stability. Besides appropriate use of antibacterial agents, initial debridement with implant retention is usually performed for both goals, resulting in implant retention rates of 40 to 100%.[1–9] However, those two aims are often contradictory; implant retention may prevent bacterial eradication because of the presence of biofilm on metal hardware, which diminishes the effect of antibiotics.[10] Yet if the hardware is removed before graft fusion, in an attempt at infection

control, the result may be spinal instability, causing clinical symptoms of backache, radicular pain, or neurologic deficits. When surgeons are dealing with infection management and spinal stability, they should not routinely choose debridement with implant retention. Instead, they must take into consideration patients' characteristics, such as immunity affected by physical strength or comorbidities, bone quality affected by age or initial spinal pathologies, original stability of the instrumented construction affected by surgical procedures, and the severity of wound contamination. To clarify the clinical features of SSI treatment after instrumented spinal surgeries, we performed a retrospective multicenter study.

Materials and Methods

We conducted a retrospective multicenter survey of 20 hospitals affiliated with the Department of Orthopaedic Surgery of Osaka University Graduate School of Medicine, after first obtaining institutional review board approval. Between 2004 and 2008, 37 cases of deep SSI were identified among 3,462 instrumented spinal surgeries, a rate of 1.1%. We defined deep SSI according to the criteria of the Centers for Disease Control and Prevention,[11] as a condition resulting in an abscess or other evidence of infection in deep soft tissue muscle and fascia, and we confirmed the presence of deep SSI by reoperation or by histopathologic or radiologic investigation. The total number of instrumented surgeries and surgeries for SSI was obtained from the surgical records of each hospital. To match the criteria for deep SSI, we selected cases by reviewing surgical records and excluding superficial SSI, which was infection involving only the skin and subcutaneous tissue, and delayed wound healing without signs of infection. We reviewed the details and treatment course for each patient. Deep SSI was present in 37 patients (26 men and 11 women), who had a mean age of 65 years (range, 4 to 87 years). Because this was a retrospective multicenter study, there was no common treatment protocol among the 20 institutes, and treatment in each case was conducted by a surgeon in consultation with an infectious-disease specialist. However, the basic strategy of surgical debridement and appropriate antibiotic therapy was common among all institutions. Depending on the bacteria identified, empirical or definitive intravenous antibacterial administration was continued until several weeks after a normalized level (< 0.3 mg/dL) of C-reactive protein (CRP) had been regained, and then oral antibiotic therapy was administered for several months. The decision between implant retention and removal during reoperation depended on the surgeon's judgment, taking into consideration implant loosening, contamination of the surgical site, and the patient's ability to endure multiple surgeries versus a longer treatment period. Deep SSI was diagnosed at an average of 32 days (range, 5 to 117 days) after the initial operation because of wound dehiscence or purulent drainage (47%), high fever (>38°C) or abnormal laboratory findings (31%), needle-aspirated discharge (17%), or for other reasons (5%).

Organisms isolated from cultured wound exudate, surgical-site tissue, or blood included methicillin-resistant Staphylococcus aureus ([MRSA] $n = 15$; 40.5%), methicillin-resistant S. epidermidis ($n = 5$; 13.5%), S. aureus ($n = 2$; 5.4%), coagulase-negative Staphylococcus ($n = 2$; 5.4%), miscellaneous ($n = 6$; 16.2%), and undetected ($n = 9$; 24%); there were two polymicrobial infections. Spinal pathologies at the initial operation were degenerative thoracolumbar disease (spinal canal stenosis, herniated disc, and spondylolisthesis, called the DEG group; $n = 15$), osteoporotic vertebral collapse (the OVC group; $n = 10$), metastatic spinal tumor ($n = 5$), spinal trauma ($n = 2$), atlantoaxial disorders ($n = 2$), rheumatoid spinal disorders ($n = 2$), and tubercular spondylitis ($n = 1$). Because of the extent of their physical debilitation, 5 patients with metastatic spinal tumors, 1 patient with spinal trauma that complicated multiple organ injuries, and 1 patient with tuberculosis were excluded from our study. Another patient with revised lumbosacral fusion for adjacent segment collapse after a long thoracolumbar posterior fusion for rheumatoid spondylitis was also excluded, because of representing too small a number to be categorized separately. This left 29 patients (20 men and 9 women) for our study population.

To facilitate simple analysis of various backgrounds, we classified patients with deep SSI into three groups according to combinations of similar initial diagnoses, surgical sites, and procedures. In addition to the DEG group ($n = 15$) and OVC group ($n = 10$), there was the cervical group, which consisted of four patients with cervical disorders (one case each of cervical fracture, atlantoaxial rotatory fixation, atlantoaxial subluxation, and rheumatoid cervical spondylitis). Details about patients such as age, sex, preoperative American Society of Anesthesiologists physical status,[12] presence of diabetes mellitus, history of previous operations, initial surgical procedures, number of fused segments, artificial graft materials used, and implants are listed in ►Table 1.

For all patients, we recorded the time required to achieve a normalized CRP level (< 0.30 mg/dL), indicating infection cure; length of additional hospital stay after the infection was resolved; the duration of the follow-up period; the percentage of those with clinical symptoms who were ambulatory before surgery versus at final follow-up examination (the ambulatory rate); and final radiologic fusion status. We regarded radiographic evidence of a continuously integrated graft or lack of segmental motion on lateral flexion–extension radiographs as documentation of fusion. We also determined the implant retention rate and analyzed the details of additional operations for deep SSI. Besides patients' backgrounds, in each group we looked for infectious conditions that could affect implant management, such as onset time of deep SSI, maximum daily body temperatures, laboratory findings for CRP level and white blood cell (WBC) count at the time of deep SSI diagnosis, detection rate for purulent discharge by spontaneous wound breakdown or needle aspiration, and identification rate for bacterial organisms, especially MRSA. Onset time was defined as the period from initial surgery to deep SSI diagnosis and was classified as either early infection (occurring within 30 days after surgery) or late infection (occurring > 30 days after surgery).[13,14]

Data are presented as means ± standard deviation, and p values of < 0.05 were considered to indicate statistical

Table 1 Background data and details of initial surgery

	Group			
	DEG	OVC	Cervical	*p* value
n	15	10	4	
Age (y)	63 ± 14	77 ± 6	39 ± 30	<0.01[a]
Sex (M:F)	12:3	5:5	3:1	
ASA status				
1	5		1	
2	10	6	3	
3		4		
No. of patients with diabetes mellitus	2	1	0	
No. of patients with history of previous operations	4	1	0	
Initial surgery	PLIF: 13	PSF with anterior graft: 7	PSF: 4	
	TLIF: 1	ASF + PSF: 1		
	PTIF: 1	PSO: 1		
		ASF: 1		
No. of fused segments	1.1 ± 0.3	3.9 ± 1.5	2.0 ± 0.8	
Artificial graft materials				
Cage	13	2		
HA		5		
Implant				
Pedicle screw system	14	9		
Spinous process plating system	1			
Anterior screw-rod system		1		
Posterior screw-rod system			3	
Sublaminar wiring			1	

Abbreviations: ASA, American Society of Anesthesiologists; ASF, anterior spinal fusion; Cervical, cervical disorders; DEG, degenerative thoracolumbar disease; HA, hydroxyapatite; OVC, osteoporotic vertebral collapse; PLIF, posterior lumbar interbody fusion; PSF, posterior spinal fusion; PSO, pedicle subtraction osteotomy; PTIF, posterior thoracic interbody fusion; TLIF, transforaminal lumbar interbody fusion.
Note: ASA physical status classification: 1, healthy patient; 2, patient with mild systemic disease; 3, patient with severe systemic disease that is not incapacitating; 4, patient with an incapacitating systemic disease that is a constant threat to life; 5, moribund patient.
[a]*p* value indicates significance in three groups, as shown by the Kruskal-Wallis test.

significance. Statistical analyses were performed by means of the Kruskal-Wallis test, Mann-Whitney *U* test, chi-square independence test, and Fisher exact test, using SPSS statistical software (version 18.0; IBM, Armonk, New York, United States).

Results

Infections resolved in all patients, with no recurrence during the follow-up period. The time elapsed from deep SSI diagnosis to infection resolution in the DEG group, the OVC group, and the cervical group was a mean of 106 ± 135 days, 77 ± 79 days, and 44 ± 23 days, followed by mean additional hospital stays of 48 ± 74 days, 131 ± 142 days, and 28 ± 11 days, respectively. There was no significant difference in length of treatment period among three groups. The respective mean duration of follow-up after discharge for each group was 18.7 ± 15.4 months (range, 0 to 48.5 months),

5.7 ± 11.2 months (range, 0 to 36.2 months), and 48.3 ± 19.6 months (range, 27 to 70.3 months). The ambulatory rate was unchanged after surgery for the DEG group, whereas 40% of the OVC group and 75% of the cervical group were ambulatory at the final follow-up evaluation. The fusion rate was 73% in the DEG group, 30% in the OVC group, and 100% in the cervical group (►**Table 2**). Implant retention rates and details of additional operations for SSI treatment appear in ►**Table 3**. Patients in the DEG group tended to require complex procedures, such as cage removal or anterior debridement and fusion, followed by continuous wound irrigation or delayed wound closure. In the DEG group, the 80% implant retention rate at the first reoperation declined to 40% by the final follow-up evaluation. In the OVC group, implant retention was attempted for only 20% of patients at the first trial, resulting in a final retention rate of 0%. The cervical group had an implant retention rate of 100%. There was a significant difference in final implant retention among the three groups

Table 2 Clinical symptoms in relation to ambulatory rate and radiologic evidence of fusion

	Group		
	DEG	OVC	Cervical
n	15	10	4
Preoperative ambulatory rate (%)	93	10	50
Postoperative ambulatory rate (%)	93	40	75
Fusion rate (%)	73	30	100

Abbreviations: Cervical, cervical disorders; DEG, degenerative thoraco-lumbar disease; OVC, osteoporotic vertebral collapse.

(►**Table 3**). There was no significant difference in onset time of deep SSI or parameters of infections, as shown in ►**Table 4**.

To study factors affecting implant retention, we investigated the DEG group further; it had a final implant retention rate of 40%. We analyzed several parameters for early infections in six patients whose implants were retained and in six whose implants were removed, excluding three patients with late infection whose implants were removed. As a consequence, we found a lower maximum daily body temperature at the time of SSI diagnosis ($p = 0.01$), lower WBC counts at the time of deep SSI diagnosis ($p = 0.04$), and a lower detection rate for purulent wound discharge ($p < 0.01$) in the implant retention group with early onset infection (►**Table 5**).

Table 3 Details of surgical procedures for surgical-site infection and implant retention rate

	Group			
	DEG	OVC	Cervical	*p* value
n	15	10	4	
Reoperation (times)	1.9 ± 1.3	1.5 ± 0.7	1	0.12[a]
Cage removal	10	0	0	
Revision ASF	3	0	0	
Continuous wound irrigation	4	0	1	
Delayed wound closure	2	0	0	
Vancomycin cement beads	0	1	0	
Implant retention rate at first reoperation (%)	80	20	100	
Implant retention rate at final evaluation (%)	40	0	100	<0.01[b]

Abbreviations: ASF, anterior spinal fusion; Cervical, cervical disorders; DEG, degenerative thoracolumbar disease; OVC, osteoporotic vertebral collapse.
[a]Significant difference in three groups, as indicated by the Kruskal-Wallis test.
[b]Significant difference in three groups, as indicated by the chi-square independence test.

Table 4 Details of deep SSI

	Group			
	DEG	OVC	Cervical	*p* value
n	15	10	4	
Onset time of deep SSI (d)	24 ± 30	51 ± 36	24 ± 16	0.82[a]
Early (<30 d)	12	4	2	
Late (>30 d)	3	6	2	0.11[b]
Parameters at diagnosis of deep SSI				
Maximum daily body temperature (°C)	38.3 ± 0.8	38.5 ± 0.9	38.2 ± 1.4	0.82[a]
CRP (mg/dL)	11.7 ± 10.6	14.0 ± 7.1	9.6 ± 8.5	0.39[a]
WBC count (mm^3)	$10,406 \pm 4,635$	$12,649 \pm 7,224$	$9,615 \pm 5,300$	0.38[a]
Detection rate of purulent discharge (%)	53	70	100	0.20[b]
Bacterial identification rate (%)	66	80	100	0.36[b]
MRS identification rate (%)	46	60	100	0.16[b]

Abbreviations: Cervical, cervical disorders; CRP, C-reactive protein; DEG, degenerative thoracolumbar disease; MRS, methicillin-resistant *Staphylococcus*; OVC, osteoporotic vertebral collapse; SSI, surgical-site infection; WBC, white blood cell.
[a]Significant difference in three groups, as indicated by the Kruskal-Wallis test.
[b]Significant difference in three groups, as indicated by the chi-square independence test.

Table 5 Analysis of data on implant retention in DEG group with early onset SSI

	Group		
	Implant retention	Implant removal	p value
n	6	6	
Diabetes mellitus	0	2	0.22[a]
History of previous surgery	1	2	0.50[a]
Maximum daily body temperature (°C)	37.9 ± 0.4	38.9 ± 0.6	0.01[b]
CRP (mg/dL)	9.1 ± 6.1	16.8 ± 13.4	0.20[b]
WBC count (mm^3)	8,633 ± 2,248	13,883 ± 4,577	0.04[b]
Detection rate of purulent discharge (%)	16	100	<0.01[a]
Bacterial identification rate (%)	50	83	0.27[a]
MRS identification rate (%)	16	67	0.12[a]

Abbreviations: CRP, C-reactive protein; DEG, degenerative thoracolumbar disease; MRS, methicillin-resistant *Staphylococcus*; SSI, surgical-site infection; WBC, white blood cell.
[a]Fisher exact test.
[b]Mann-Whitney *U* test.

Discussion

Diabetes mellitus, smoking, previous surgery, fusion, longer duration of surgery, and poor general or functional status have been reported as risk factors for SSI after spinal surgery.[1,7,15–17] In addition to providing prophylaxis, it is important to know how to treat deep SSI, especially because it has not been definitively established whether it is best to remove implants or instead leave them in place after the diagnosis of deep SSI. The clinical practice guidelines for the treatment of MRSA infections recommend device removal whenever feasible in late-onset spinal-implant infections (>30 days after implant placement).[14] It has been reported that in late infections occurring several months or years after the initial operation, implant removal is preferable because of documented bony fusion or difficulty in resolving infection.[9,13,18] In early infections, debridement with implant retention is ideal and should be attempted initially,[13,14] but debridement does not always ensure cure. Rates of successful implant retention of 92 to 100% have been reported by authors who used multiple debridements in combination with continuous irrigation, antibiotic-impregnated cement beads, or secondary wound closure,[2,4,5,7,8] although hardware removal after multiple debridements was sometimes required.[19] Ho et al reported that there is an almost 50% chance that infection will remain if all spinal implants are not removed in posterior scoliosis surgery.[3]

To investigate the difference in the treatment course according to patients' backgrounds, we classified 29 patients into three groups by similar spinal pathology, surgical site, and procedures and then analyzed their treatment period, implant retention rates, and parameters about infection. The implant retention rates were different in each group, and we could not find any differences in treatment period and any of the parameters listed in ►**Table 4**, except for age (►**Table 1**). In the OVC group, which was characterized by advanced age,

hardware removal was done at the first reoperation in two patients with early infection, because of screw loosening, whereas in two other patients with early infection, implant retention was attempted but failed, with removal being required later for infection treatment. In six patients with late infection, hardware was initially removed because of pedicle screw loosening due to preexisting osteoporosis as well as deep infection. However, the cervical group, 50% of which had late infections, had an implant retention rate of 100%. In the cervical group in our series, patients initially received bone grafts only on laminae or facet joints, so the lack of a need for interbody debridement might be advantageous in infection management with implant retention. Implant retention was successful in 6 of 12 patients with early infections in the DEG group after single or multiple episodes of debridement, with or without cage replacement, using autologous iliac bone grafting and a posterior approach. In the other five patients, implant removal was eventually required, along with switching cages to autograft in some cases, via anterior or posterior approaches, although implant retention was attempted during the first reoperation. In another patient with early infection, implant removal was initially performed. In late infection in three cases, implant retention was attempted in one patient during the first reoperation, but the implant failed later; in the other two patients, implants were removed during the first reoperation. Insufficient eradication of infection in the presence of hardware was the reason for implant removal in the DEG group.

Immunocompromise predisposed by comorbidities could be associated with implant salvage. Patients with diabetes mellitus were not able to retain their implants in our series, although this finding was not statistically significant. Kowalski et al conducted a univariate analysis of risk factors for treatment failure in patients with early and late-onset spinal infections and found no significance for immunocompromise, but they did find that systemic malignancy and prior

radiation therapy posed significant risks.[13] It was not applicable to our series because we excluded malignancy, but their other finding that treatment failed in all patients with diabetes mellitus or hepatic failure seemed to be reasonable. We hypothesized that in addition to time of onset to infection, immune status, and initial spinal pathologies, the degree of wound contamination was associated with implant salvage, because of the lower detection rate of purulent wound discharge, lower maximum daily body temperature, and lower WBC count in the implant retention DEG group with early onset infection. We speculated that one of the reasons for implant salvage was early recognition of SSI. In patients who retained their implants, SSI might be suspected and diagnosed at an early stage of bacterial incubation, before infection had reached an advanced enough stage to produce an abscess that would break down the overlying skin or could be aspirated or to cause a high body temperature. Prompt diagnosis at an initial stage with a low intensity of infection could reduce the necessity of implant removal.

Three groups have investigated whether laboratory data are helpful in the early detection of wound infection.[20–22] With the range of postoperative CRP peak ranging from day 4 to day 7, all three groups agreed that reelevation of CRP level or an increase in CRP after the peak day suggested the possibility of SSI. However, the usefulness of CRP level in this setting is limited because it also reflects other systemic infections or inflammations with unknown causes. Kang et al. investigated serial postoperative changes in CRP in 348 patients, resulting in 16 cases of abnormal CRP response.[21] There were 5 cases of infection related to spinal surgery, 3 cases of infection in the digestive or urinary tract, and 8 cases of unknown cause. Laboratory findings provide clues to deep SSI, but they are less reliable in diagnosis without corresponding wound symptoms.

Our study had several limitations. Because it was a retrospective multicenter survey—which provided the benefit of large sample size—evaluations of clinical outcomes from medical records were not standardized, especially regarding pain assessment. The only comparable symptoms were preoperative and final walking status. In the OVC group and the cervical group, in which the chief problem was inability to walk because of leg paresis, clinical outcomes measured by ambulation would be useful in assessing the benefit of deep SSI treatment. However, in the DEG group, in which the chief problem was back or leg pain and in which 93% were ambulatory before surgery, an unchanged ambulatory rate without pain assessment did not reflect the clinical effectiveness of deep SSI treatment. Another limitation was small sample size resulting from a low infection rate of 1.1%, although the sample was collected from 3,462 cases in multiple institutions. Regarding weak statistical power, we warrant the homogeneity of patients by categorization into multiple groups of similar backgrounds. Although our numbers were relatively small, the homogenized DEG group of 15 and the OVC group of 10 provided convincing documentation of implant handling in each group. Although the size of the cervical group was also too small for extracting a conclusion regarding significance, results of the group are presented in this report because they illustrate

the clinical features of our SSI series and because a rate of 100% implant retention is informative.

The final implant retention rates were 40% in the DEG group, 0% in the OVC group, and 100% in the cervical group, indicating that implant retention was affected by spinal pathologies. In the OVC group, implant salvage and good clinical outcomes were unobtainable because of preexisting osteoporosis and preoperative symptoms of paraplegia. Implant removal at first reoperation should be considered for the purposes of avoiding multiple operations in the elderly and resolving infection in the shortest possible time. In our cervical group without interbody procedures, it was possible to attempt implant retention.

In the DEG group with early onset infection, it was possible to attempt initial debridement and to leave implants in place, keeping in mind that the success rate was 50%. For this population, in whom screws are not expected to be loose, we believe the key to success is early suspicion of SSI. Our analysis of early infection in the DEG group suggests a relationship between implant retention and intensity of infection, especially when purulent discharge is present. Early debridement before detection of purulent discharge could decrease the chances of implant removal. If reelevated laboratory markers after postoperative day 4 to day 7 or fever of ~38°C is observed, surgeons should immediately suspect deep SSI. However, without skin breakdown or spontaneous drainage, a diagnosis of SSI will not be confirmed, so surgeons should investigate other possible causes of inflammation, such as pneumonia or urinary or digestive tract infection, by the use of imaging studies or bacterial cultures. If fever origin is not identified even after sufficient examination, we urge that surgeons do not hesitate to perform needle aspiration of the surgical site or exploratory surgery to confirm diagnosis before drainage appears, instead of taking a wait-and-see approach. Until a reliable, less invasive detection method for early deep SSI is available, exploratory surgery for early debridement is the only way to reduce the risk of implant removal.

Conclusions

The final implant retention rates of 40% in our DEG group, 0% in our OVC group, and 100% in our cervical group represent clinical features of implant handling in each group. Besides infection onset time, implant retention may be affected by initial spinal pathology. In DEG with early infection, prompt debridement at a stage of lower intensity of infection before drainage may be advantageous to implant salvage.

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Disclosures

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